THE RELATION OF MECHANICAL DISTENTION TO THE ETIOLOGY OF APPENDICITIS.[1]

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IN a study of appendicitis, four conditions point the way to its etiology. 1. The constriction; 2. The concretion; 3. The pathogenic germs, always present; and 4. The distention, observed in early operations.

These have all been repeatedly commented upon in searching for the cause. One other necessary condition is the hydrostatic pressure in the distended appendix. Pressure upon the blood-vessels in the interior of the appendix lowers the resistance and the germs do their work. If continued, it obliterates blood-vessels and produces necrosis and gangrene. Many attempts have been made to explain the vascular changes by trauma or pressure from concretions, foreign bodies, or kinking or torsion; but the fact that the fluid can produce pressure upon blood-vessels as well as solid bodies has been overlooked. What will furnish a more simply or satisfactory explanation than this fluid distention?

The appendix is essentially composed of a hard, resisting, muscular wall enclosing a mucosa and submucosa and covered with peritoneum. The blood-vessels branching in the meso-appendix pierce the wall in many places and, *ramifying the submucosa,* supply the mucosa. The latter is rich in lymphoid tissue. The lumen contains mucus, fæcal matter, remains of food, and a rich supply of bacteria.

Given a constriction in any part of the lumen of the appendix with a concretion behind it (passing the mode of their formation for the present), let us briefly trace the process of

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distention and inflammation. In any one of a number of ways the concretion closes the opening in the constriction. Secretion from its numerous glands may furnish the first fluid in this closed cavity, or there may be a considerable quantity of fluid already present at the moment of closure; it may be sufficient to produce decided pressure immediately.

There are now three different fluids involved in this process, all subject to different pressures and separated by only the filmiest membranes.

1. The afferent vessels (the arteries) carrying fluid into the appendix at a pressure of, say, 125 millimetres mercury;

2. The efferent vessels (the veins and lymphatics) carrying fluid out of the appendix at from nothing to ten millimetres;

3. The fluid in the occluded cavity subjected to a pressure of, say, twenty-five millimetres, may be anywhere from ten to 100.

Hydraulics and dialysis tell us what follows in the struggle for hydraulic equilibrium. The efferent vessels which are within the resisting wall of the appendix will be more or less occluded by the greater pressure in the cavity. The capillaries responding to the obstruction beyond dilate and allow serum to escape; lymphatic drainage is blocked, effusion results. Every atom of fluid which remains within the wall of the cavity adds its pressure to that already present and causes greater obstruction to the efferent current. The arteries keep on pumping in the fluid at 125 millimetres; effusion continues, and the pressure in the cavity rises and continues to rise until it equals that in the arteries, and these pressures are balanced at 125 millimetres, or whatever the arterial tension may be.

Now, if this occurred in perfectly sterile surroundings, this balance might be maintained for some time without material harm; enough fluid would filter through to maintain life in the cell. But our cavity is far from sterile; that innumerable numbers and varieties of germs make this their home has been repeatedly demonstrated. With the circulation impeded, the tissue cells, deprived of their supply of food and oxygen, offer little resistance to the attack of germs. The leucocytes are prevented from reaching the point of assault; the mouth of every follicle and gland and lymphatic is opened wider with every increase of pressure—doubtless microscopic trauma occurs. What more perfect conditions for "infection" can be conceived?

With the increase of pressure and the infection infiltration, complete strangulation, necrosis, and gangrene follow. Our cavity is now a real abscess cavity, subject to the same laws of hydraulics, circulatory interference from hydrostatic pressure, infection infiltration, necrosis, and advance in the direction of least resistance.

The comparison and detailed study of these processes in an abscess and in the appendix open an attractive field, but I cannot take time for that now.

Assuming that the obstruction does not give way, necrosis appears; first in the most vulnerable portions; thin places in the wall; parts having the poorest circulation or those affected by the most virulent infection. These break down and we have a perforation. Or if the pressure and infection are about equal in all parts of the wall, or of sufficient intensity or character, gangrene of the entire enclosed cavity will result.

Time and again such appendices have been removed within twenty-four to forty-eight hours of the onset of the first symptoms; often they are so distended that the surgeon fears they will rupture during removal; often one or more black necrotic spots are in evidence, to which protective omentum has already become adherent; or the entire appendix or that portion distal to the obstruction has been found gangrenous, but still intact. This is the ideal appendix we all strive for by early operations, and fortunate is the patient who parts with it at this stage.

In my own experience, this condition of distention obtained in every case in which the time and character of the case made it possible. Surgeons generally tell of such experiences, and many promptly add, "I can see how your explanation will account for this class of cases." If for these, then why not for those in which perforation has already taken place. To be sure, the fluid has been disseminated or is confined in a local abscess, and the walls of the appendix are collapsed; possibly the entire appendix is wellnigh lost in the mass of necrotic tissue present. Still, the case found distended at thirty-six hours after onset would have been in this same collapsed condition if left until the third or fifth or eighth day.

I must put the same argument for those other cases in which the direction of least resistance drives the concretion out into the colon. Here even the concretion is lost, and the only evidence of the condition which prevailed a few hours or minutes before is the distensible cavity with inflammation *beyond the point of constriction.*

Nevertheless, is not the analogy complete? If the constriction will in time dilate sufficiently to allow the concretion to pass under the increasing pressure, that will be the direction of least resistance. The contents will be discharged into the cæcum and the injury done to the appendix will be repaired as best it can. If the infection has only begun or is of a mild character, repair will be easy, and the continuing appendicitis will be of the so-called catarrhal type. It may be so mild as to escape observation entirely, the patient never knowing that anything unusual occurred within his abdomen. If the concretion is not passed into the colon until the distention reaches a high point or until it has been a practical strangulation for some time, or if the character of the infection is very virulent, serious changes may have taken place, necrosis, even gangrene, may continue, or the infection may continue to advance through the wall of the appendix without necrosis and produce peritonitis, or it may follow any of the devious courses so inimical to appendicitis.

So far I have considered only those typical cases which contain a great amount of constriction, together with an actual concretion. Neither of these is, however, essential to the production of appendicitis nor to this explanation of its etiology. Strictly speaking, this distention is really a step in the early pathology of the disease, but, as it is the cause of the acute attack, it is indeed the cause of the illness which we call appendicitis. A number of conditions will vary the course and the final result of this obstructive process. The constriction may occur at any point in the course of the lumen from the colonic junction to within one-half inch of the distal tip. Likewise, it varies in size from the slightest narrowing to the tightest constriction. It may be only an unusually small "valve of Gerlach," or it may be a cicatrix encircling the lumen and closing it to a pin-point opening. It may be only a "kinking" of the appendix.

The obstruction need not always be a concretion. A fæcal mass of varying size and consistency may obstruct for a sufficient length of time to produce tension, vascular changes, and infection. Under these circumstances, the infection will probably be less severe. In fact, a concretion is simply the insoluble inorganic residue of fæcal masses growing by accretion, the organic matter disappearing by digestion, bacterial action, and solution. If the constriction is very tight, possibly a plug of mucus might furnish the obstruction for a slight distention, and a mild infection, the resulting congestion of the wall completing the closure.

The determination of the degree of tension necessary to produce infection opens up an interesting field for study and experimentation. The actual manner in which closure of the trap occurs must of necessity be partly left to conjecture. The most reasonable explanation is that it depends upon accidental circumstances. I have demonstrated on an appendix which contained a concretion how easy it is to force fluid into the appendix which, by its recoil, forces the concretion into the constriction, and closes the cavity by a perfect ball-valve. It seems reasonable, from the suddenness and severity of many attacks, that that does actually happen in many cases.

Anything increasing tension in the colon thus becomes responsible for the actual onset. Diarrhœa, constipation, fermentation, flatulence, trauma, athletic strain, all these have been charged with being the cause of appendicitis; and I can see how by increasing intracolonic pressure they may be the last straw which springs the trap. Often, however, I believe the closure is purely accidental. It will come on during sleep; the position of the patient may favor it. The constriction and concretion may be ready for the final tragedy for months or years, and only await the accidental closure to bring on an attack. In fact, they are often found post-mortem after death from other causes.

It should be remembered that the concretion forms in the very mouth of the constriction, being moulded to a perfect fit by the frequent attempts of the appendix to empty itself. Still, it may for a long time remain a poor fit, and the moment pressure increases the valve leaks, the concretion falls back into its bed, and the attack is postponed. The same is true of fæcal masses.

No doubt one cause of closure is the attempt of the appendix to empty itself by the contraction of its muscular fibres, the presence of which is no longer a matter of doubt. Once closed, the appendix continues these attempts to empty itself, producing appendicular colic. This is to my mind the most reasonable explanation of the first colicky pain which is the most characteristic symptom of the onset of the disease. This " spasm," originating in the appendix, spreads to the adjoining colon and ileum, and is in some cases responsible for diarrhœa during the first few minutes or hours of the attack.

Rubin, in the *Journal of the American Medical Association*, Vol. xliii, No. 18, demonstrates how flatulence favors the entrance of colonic contents into the appendix. In so far as it does so, it favors the production of a plug as well as a closure of the constriction, and in that way may be a factor. After the trap is closed, the formation of gas from fermentation of the enclosed contents sometimes adds rapidly to the existing pressure.

R. Abbe[1] has beautifully demonstrated the formation of strictures from the ulceration following an attack of appendicitis, several often occurring in the same appendix. That all narrowing of the appendicular lumen is thus produced does not however follow. Many of the irregularities of the appendix are congenital. The club-shaped, long, narrow, or short thick appendix, the kinks, the short mesentery, and numerous other constricting conditions are simply accidental and congenital, the simple result of the degeneration of a rudimentary organ. Hence, a person is destined to have appendicitis some time during his life or not, according to whether he is born with a respectable appendix or not. And the pronounced attack is most likely to occur during the period of full development of these irregularities, not far from the period of adolescence.

These irregularities are often hereditary, like all other minute anatomical peculiarities, and that explains the hereditary features of the disease. I have seen it occur in two brothers and one sister within two years. The first attacks are usually mild, and doubtless occur during childhood, and are constantly overlooked both at that time and later in the history. What boy of ten to fourteen would think of remembering such little "belly-aches"?

The natural irregularity of the lumen retards the passage of a fæcal plug sufficient to allow a moderate distention to take place. It takes but little pressure to retard the venous and lymphatic current, lower the resistance to germs, to open the follicles and crypts of the appendix, perhaps produce minute traumatisms, and inaugurate a mild infection before the lumen again becomes patulous. This infection by infiltration, ulceration, and cicatrization tightens the stricture, and often produces others. How often do clinical histories report several such mild attacks months and years preceding the stormy one which ends in complete occlusion and resulting perforation.

Occasionally a distended appendix is found which presents no gross evidence of active inflammation, or, if it does, it is very mild. This occurs as a result of an attack of appendicitis which has run its course. In the light of these studies, I would explain these by saying they are a result of the process of resolution, the infection being mastered by the tissues, and the lumen occluded by adhesive and cicatrical processes; a cavity is left which becomes or remains distended by the hydraulic processes I have described, but as it contains no germs it produces no infection.

Sometimes a small cavity becomes permanently occluded; the few germs which it contains produce a mild infection and slight symptoms of appendicitis, but it is so mild that the germs are killed and the infection overcome without a perforation. The distention thus remaining may then continue indefinitely.

That this is indeed the explanation of the nature of appendicitis is emphasized by the following facts:

It is not sufficient to say appendicitis is due to germs which happen to lodge in the appendix as we can of most infectious and contagious diseases. No idiopathic germ can be charged with being the etiologic factor. We find no germs there which could be said to be introduced into the body for this special occasion. The germs involved in the process are normally present in the appendix in health.

The suddenness of the onset in many cases can hardly be explained except on the ground of a sudden mechanical change having taken place. The sudden cessation of spontaneous pain likewise indicates the giving away of a mechanical something which to the patient himself suggests the removal of some kind of an obstruction in the abdomen.

The inflammation, until it spreads along the peritoneum, is confined to that portion of the appendix beyond the particular constriction which causes the trouble. How often we find only the distal inch involved, and the balance of the organ proximal to the constriction as healthful as ever. Again, the inflammation is evenly distributed over that entire portion so involved, and that very early in the case, before it could have time to spread by continuity.

The frequency of necrosis and gangrene indicates mechanical obstruction. Simple infection does not do this except in abscess formation. The only abscess early in appendicitis is such as I have described occupying the lumen itself.

Then, why is the necrosis or gangrene confined to the portion beyond the stricture, unless that stricture is to blame for it? If the necrosis is due to infiltration pressure following simple infection, why should it be content with a simple perforation? Why does it not spread to the balance of the appendix and the colon, the omentum or mesentery? Often we find a small, simple abscess just outside of the perforation with an infection so mild that it is arrested right there. Would such mild infection produce necrosis from pressure infiltration? Why, then, do not we have necrosis of other portions of the alimentary canal?

Almost invariably the stricture or kink will be found prominent in proportion to the severity of the attack. In mild cases comparatively little stricture will be found.

To practically test the effect of tension upon this problem, I made a series of experiments on dogs. The appendix was ligated subperitoneally at its juncture with the colon. I am told that, strictly speaking, the dog has no appendix, that it is merely an elongated colon, but in shape, position, and general anatomical characteristics it resembles the human appendix so closely that for practical purposes it is an appendix.

An aspirating needle was passed through the wall of the colon into the lumen of the appendix, and through this the appendix was distended with tap water at any pressure desired, a manometer being used for that purpose. Cultures of Staphylococcus albus and colon bacillus were introduced into the appendix in a few cases.

The experiments were detailed in the *Journal of the American Medical Association* for March 26, 1904, so I shall not repeat them here, but merely give the summary of results. There was no difficulty in producing the picture of appendicitis, but we found that the one essential condition for its production was that the organ be distended. In all cases in which this condition was complied with the results were strikingly typical of human appendicitis. The tension was varied from seventy-five to 150 millimetres mercury, and continued for two hours or more, and in all these cases a typical appendicitis followed in twenty-four or forty-eight hours.

The other experiments of ligature simply, or of injection of cultures without a simple ligature, or with it, all failed to produce any effect upon the gross appearance of the appendix in twenty-four or forty-eight hours.

I find in the literature reports of a number of attempts to produce appendicitis in animals experimentally. The more important ones were those of Roger and Josne,[2] Cappelon,[3] Beausenat,[4] Nicholayson,[5] Lipavski,[6] Roux,[7] Adrian,[8] Mühsam,[9] and Baudler.[10] Usually the attempt was made to produce infection by some irritation or traumatism of the interior of the appendix. Several workers have injected cultures of various germs into the appendix. General infection through the blood was tried. After injecting virulent cultures of bacteria into the appendix and adding considerable traumatism, a limited infiltration is reported, but nowhere do I find a report of that general inflammation of all the coats of the appendix which gives us the picture which we call appendicitis.

In order to produce any results, the circulation must be retarded by distention and consequent pressure, and when this is done the germs normally present in the appendix promptly produce infection.

The practical bearing of this view of appendicitis upon the diagnosis and treatment is very evident. Some of its most valuable hints come from a careful study of the history of pain and tenderness during the first hours of the attack.

If the distention comes from a sudden increase of intracolonic pressure produced by athletic exertion, or accident, or gastro-intestinal disturbance (forcing fluid into the appendix, which, by its recoil, forces the concretion into the constriction, closing the ball-valve), the onset will be characterized by severe pain coming on suddenly. If the occluding mass is small or pliable and is forced out through the constriction by the increasing distention, there will be a sudden cessation of acute pain. If the occlusion is permanent (that is, if the plug is too large to pass, too hard to be compressed, and fits too accurately to allow of a leak), distention will increase until the circulation is arrested, and necrosis or gangrene will follow. After a time the nerve endings involved in that process will fail to report pain and the patient feels relieved, but we know it is only the lull before the storm, which breaks out with redoubled fury when the gangrenous tissues give way and allow the pent-up flames of infection to burst out upon the peritoneum.

I believe that in many cases we can diagnose these different conditions accurately by carefully studying the history. If I get a clear history of acute, colicky pain continuing less than an hour, or even two hours, with a fairly sudden cessation, I feel confident that the obstruction has given way, and that only a so-called catarrhal appendicitis is to be expected. There will still be pain upon moving about or from rolling of intestines, and there will be tenderness upon pressure, but no more acute, spontaneous pain. This means inflammation in the appendix, but relief of the distention and strangulation.

Again, when the pain has been severe for several hours, there has been frequent vomiting and perhaps two or three attempts at stool; there can be no practical doubt that the distention will not be relieved through the anatomical exit into the colon, but by rupture of the appendix, following necrosis or gangrene.

If this acute pain gradually diminishes after six or twelve hours, it is quite certain that considerable gangrene is present, usually complete gangrene of the appendix beyond the constriction.

The sudden reappearance of severe pain twenty-four to twenty-eight hours after beginning of the attack means peritonitis following. There are, of course, cases on the border lines in which the actual condition cannot be known; but generally the distinction between the so-called catarrhal cases and perforative ones can be made out and the presence of absence of peritonitis can practically always be determined.

A patient of mine recently gave a typical history of recurrent attacks of mild appendicitis in one sentence. She said, " I would have severe pain for a short time, and then be ' so sore' for several days afterwards." This is the characteristic history. Severe pain while the distention and strangulation continue; relief from acute pain as the obstruction escapes, but continued tenderness from the inflammation meanwhile established, by the germs driven into the exsanguinated tissues.

The treatment as suggested by this theory may, in the first stage, be likened to that of strangulated hernia. The pressure upon the blood-vessels is practically a strangulation. If the strangulation is not promptly relieved, necrosis or gangrene will follow. We say, "Don't allow the sun to set upon a strangulated hernia." We may say with equal propriety, "Don't allow the sun to set upon a strangulated appendix." Could we always know what strangulation was or was not relieved, the indication would always be clear. Unfortunately, we cannot *always* be certain. Still, study, from this stand-point of distention as a cause, throws a flood of light upon the subject, and failure to make this diagnosis should be rare. Unfortunately, again, patients so commonly fail to call the surgeon during this early period, when it is simply a question of strangulation. In a large proportion of cases it is the resulting infection which claims the attention of the surgeon,—so often already a peritonitis. If the pain has been slight and of short duration, and only slight tenderness remains, it is safe to say that the case needs no treatment from that attack. Of course, he will probably have further attacks, and need his appendix removed on that ground. The appendicular colic is evidence of a constriction. A plug of fæces or a fæcal concretion will reform and sooner or later produce another attack. But for the present he is safe with a simple catarrhal appendicitis.

The possibility of dislodging this plug either into or out of the appendix by some form of taxis or massage occurs to me, but I have had no opportunity of testing it. As a matter of fact, we are rarely called early enough to apply it (within three or four hours), and many anatomical difficulties present themselves.

Operation, if performed at all, must be done before perforation, or peritonitis, supervenes. The question of early operation should be settled early, within twelve hours if possible, almost certainly within thirty-six hours, and at least within forty-eight hours of the onset of symptoms. It is impossible to make rules by hours or by the severity of the pain. Distention advances at such varying rates and the virulence of germs varies so greatly. The weight given to the evidence derived from a careful study of time and pain must depend upon the judgment and experience of the surgeon. It is comparative evidence and cannot be tested by a fixed measure. It should be only the most positive evidence that allows the case to go on with the assurance that only a mild attack without consequence is impending. When in doubt during these early hours, I advise operation every time. If the error is made of removing a catarrhal appendix early, no harm is done. It is even then a good riddance to the patient.

But appendicitis is treacherous, and more than time and pain must be considered. Sometimes the most serious cases have little or no pain during the first few hours. All the symptoms must be studied, of course, but always bearing in mind the probable stage of the strangulation. But, unfortunately again, we are so often called upon to consider this question of operation when we know the case is one of peritonitis rather than appendicitis only. It may be limited to a small portion of the peritoneum, may be a walled-off abscess, but, nevertheless, peritonitis. The removal of all the infected tissue is now out of the question, and to my mind Ochsner has conclusively proven that a much larger percentage of such cases get well if left alone than if meddled with by operation. A certain number will die with any kind of treatment, but my personal experience with both forms or procedure is overwhelmingly on Ochsner's side of this exceedingly serious controversy.

In the presence of acute peritonitis a focus of acute infection is certain to remain after operation, and the danger of its spreading is immensely increased by the intestinal paralysis, the interference with the normal secretions of the peritoneum, and the removal of the barriers already thrown up by nature's protective forces.

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